Control of airborne contagion of tuberculosis[1]

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THE CONTROL of an infectious disease may be achieved by effectively increasing the resistance of a population to the causative agent of the disease or by preventing the penetration of the virulent agent into the susceptible hosts. Thus, smallpox is eliminated by vaccination and typhoid fever by sanitary removal of pathogenic bacteria from water and milk. In tuberculosis, while some success has attended the use of BCG vaccination it is far from complete. Until such time as a universally successful immunizing agent is developed, the chief protection against the disease must be sought in the prevention of invasion of the body by the tubercle bacillus. Therefore, knowledge of the mode of infection in this disease is of fundamental importance in its control.

The old concept that infectious diseases are caused by gaseous agents, miasma, disseminated by air was eliminated by modern bacteriology. In the past generation it has been widely held that, barring diseases conveyed by insect vectors, such as malaria, direct contact with infected individuals or with carriers is the most usual way of transmission of infections. Yet the eminent exponent of this theory of contagion, Chapin(1), felt that, of all communicable diseases, tuberculosis is most likely airborne. That tuberculosis can be acquired by ingestion of milk contaminated with tubercle bacilli is beyond question. The practical elimination of bovine tuberculosis of man in the United States in the past 25 years that has followed the eradication of tuberculosis from our herds (2) and the widespread simultaneous use of pasteurized milk attest to the efficacy of this method of eliminating tubercle bacilli from our food and the control of tuberculosis by the alimentary portal. However, pulmonary tuberculosis of the human type still kills more individuals in the most productive period of youth and early adult life than any other disease. This is due primarily to our inability to prevent the entrance of tubercle bacilli into the lungs.

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Origin

How is pulmonary tuberculosis acquired? It is often said that tuberculosis originates from the aspiration of droplets containing tubercle bacilli which are expelled into the air by coughing individuals. It must be emphasized at once that there is much circumstantial evidence to indicate that coarse droplets—those larger than 0.2 millimeters in diameter—are in themselves incapable of causing pulmonary tuberculosis(3). Such large particles quickly settle to the floor and, if they happen to be aspirated by an exposed individual during this short flight, they are arrested at once in the nasal and upper respiratory passages. Tuberculosis rarely if ever originates in the nose, throat, or even in the upper bronchi.

Pulmonary tuberculosis usually has its origin deep in the lung parenchyma, in the terminal bronchioles and alveoli of the lung. The anatomical and physiological characteristics of the lung, and the known behavior of particles suspended in air, suggest that only extremely minute and totally invisible particles, not larger than several μ or a few thousandths of a millimeter in diameter can penetrate this portion of the lung. It was demonstrated by Wells(4) that droplets less than 0.1 millimeter in diameter on entering the air evaporate instantaneously and the solid bacteria or other agents of disease contained therein, the *droplet nuclei*, remain suspended in the air and are wafted about by air currents like smoke. It is these particles or nuclei that penetrate deep into the lung with the inhaled air. When trapped by settling in the terminal lung passages they may cause pulmonary tuberculosis. As stated, these are but general concepts which must be confirmed by experimental evidence.

That air is actually a natural vehicle of the contagion of tuberculosis in animals was demonstrated in the following manner (5). If normal guinea pigs or rabbits are placed in individual cages in a room housing tuberculous animals, the exposed individuals acquire a tuberculosis which originates deep in the lung parenchyma and is accompanied by massive tuberculous involvement of the tracheobronchial nodes draining the pulmonary portal of entry. The mesenteric or cervical nodes, draining the alimentary portal, are not affected. As is well known, tuberculosis characteristically leaves the traces of its progression in the body by the involvement of the nodes draining the portal of entry. It is clear therefore that the disease acquired by these animals is of respiratory origin. Whether the exposed individuals were situated in immediate proximity to tuberculous roommates or at a considerable distance from them, the incidence of the tuberculosis was the same. The infectious agent was apparently uniformly distributed in the room. Since the tuberculosis originated deep in the lung parenchyma through inspired air it is evident that the contagion was airborne.

That droplet nuclei actually cause pulmonary tuberculosis as seen in man was shown by the following experiment(6). A suspension of tubercle bacilli was sprayed from a flask into a mixing chamber. After all coarse droplets from this spray had settled, the invisible droplet nuclei were carried by air suction into a long pipe(7) and thence into a chamber where rabbits were exposed. After some 4 weeks the exposed animals were killed and it was found that the number of primary tubercles that had developed in the lung was proportional to the number of droplet nuclei contained in the air breathed by the rabbits. These primary tubercles corresponded to the primary foci seen in the lungs of human tuberculosis.

Since it has been known for a long time that ultraviolet rays are bactericidal, tubercle bacilli in the form of droplet nuclei suspended in the air were exposed to the rays(6). It was found that a few seconds' exposure was sufficient to kill all but a few bacilli suspended in the air.

If inhaled droplet nuclei are the cause of naturally acquired pulmonary tuberculosis, and if ultraviolet light can kill these droplet nuclei suspended in the air, it would follow that natural airborne contagion of tuberculosis should be prevented by ultraviolet radiation.

Accordingly the following experiment was set up(8). A large manifold is separated

in the middle by a fine wire mesh screen. On one side of the screen is a run for artificially infected rabbits which shed tubercle bacilli in their urine. On the other side of the screen, in individual cages with open wire meshwork in back and in front, are placed normal litter mates of highly inbred rabbits of high and low inherited resistance to tuberculosis. The room housing the manifold is divided by a solid partition extending from the floor to the ceiling which also divides the interior of the manifold into two equal air-tight halves. Each room contains the same number of infected sources of contagion and the same number of exposed litter mates of the resistant and the susceptible families. One room is irradiated with ultraviolet lights. The other is not irradiated. The exposed litter mates are left undisturbed in each room. The infected rabbits serving as sources of contagion are interchanged daily between the two rooms. Thus both host and parasite variables are the same in both rooms. The only difference between them is the presence or absence of ultraviolet lights.

At the end of a year, 11 of the 15 contacts in the unirradiated room died of tuberculosis. These included rabbits of high and low resistance to the disease. None of the 15 litter mates of the same genetic resistance to tuberculosis, exposed to the same contagion for the same time in the irradiated room developed tuberculosis. Thus a 73 per cent mortality from airborne tuberculosis was eliminated by ultraviolet radiation. The disease which developed in the unprotected rabbits originated as a single focus deep in the lung parenchyma and was similar to those foci found in rabbits exposed to air artificially contaminated by droplet nuclei. It is clear from this experiment that natural airborne contagion in rabbits caused by the inhalation of either droplet nuclei or microscopic, air-drifting dust particles carrying tubercle bacilli can be eliminated by the bactericidal effects of suitable ultraviolet radiation.

No clinical data are as yet available on the efficiency of ultraviolet irradiation in the prevention of human airborne tuberculosis. However, an increasing volume of observations indicate that measles, mumps, chicken pox(9), common colds, upper respiratory infections(10), as well as infections of surgical wounds in operating rooms(11) can be significantly reduced by suitable radiant disinfection of the air. Since in the experiments on rabbits cited above, the degree of contamination of the air by tubercle bacilli is very much greater than is likely to occur in human habitations, it is most probable that ultraviolet radiation will reduce airborne tuberculosis in man. There is no evidence that, if properly installed, ultraviolet lamps will cause any injurious effects on the occupants of irradiated rooms(12).

Another method of sterilizing air is by the introduction of germicidal vapors into occupied rooms. The original observations of the English workers with hypochlorites and resorcinol(13) followed by the extensive experimentation and clinical data gathered by Robertson(14) and his collaborators in this country indicate that propylene and triethylene glycols will kill various bacteria and the virus of influenza when used in the proper concentration and under suitable conditions of temperature and humidity. Several observers have found that the glycols materially reduce the incidence of upper respiratory infections in man(15). Whether these vapors are effective on tubercle bacilli drifting in air has not as yet been determined.

Many upper respiratory infections as distinguished from diseases which originate deep in the lung are conveyed by droplets and dust particles which contaminate the air for short intervals after their introduction. Bedding of patients who have hemolytic streptococci in their nasal passages is frequently contaminated by the virulent micro-organisms(16). It may be presumed that the bed clothes of tuberculous patients may likewise store tubercle bacilli. To reduce this source of contagion bedding has been treated with stable oil-in-water emulsions as a final rinse in the laundering process(17). Oil-treated bedclothes retain their ability to prevent the dissemination of dust and bacteria into the air for long periods of time. The application of oil to floors will reduce the degree of bacterial contamination of the air. There is some evidence that the use of both the above procedures may reduce the incidence of endemic respiratory infection (18). There is no cogent reason why both ultraviolet radiation and oil treatment of bed clothes and floors should not be used in hospitals for tuberculous patients, especially since the present experimental evidence does not distinguish between droplet nuclei and minute, air-drifting dust particles as agents responsible for airborne tuberculosis.

Mechanical barriers such as cubicles (19) and wide spatial separation between individuals(20) occupying the same rooms may reduce contact infection. They do not reduce the incidence of diseases caused by droplet nuclei or minute dust particles which remain suspended in the air; for the natural ventilation of dwellings soon distributes these particles uniformly throughout a given enclosed space.

This brings us to a few words on the efficacy of fabric masks in the control of airborne infection. The high-speed photography of Jennison(21) has shown that "there is great variation in the permeability of masks in common use. None of those tested was impermeable to droplets produced in violent sneezing." The ordinary two-layer gauze masks(22) permit the penetration of 23 per cent of droplet nuclei passed through them. It is noteworthy that 6-layer gauze masks if laundered 20 times become quite impermeable to bacteria and yet offer little resistance to air flow and hence to breathing. From our present state of knowledge it would seem that face masks cannot be relied on as efficient barriers to the inhalation of droplet nuclei which are the most dangerous particles from the standpoint of the origin of tuberculosis.

Summary

Pulmonary tuberculosis is largely an airborne disease. It originates from the inhalation of invisible droplet nuclei or microscopic dust particles carrying tubercle bacilli. Natural airborne contagion of tuberculosis can be prevented by suitable ultraviolet irradiation of the air. Germicidal vapors are effective against a variety of pathogenic micro-organisms and viruses. Whether they can be used in the control of airborne tuberculous contagion has not yet been determined. Nor has it been definitely established that face masks are effective barriers against droplet nuclei. The use of oil on floors and bedding materially reduces the contamination of air by bacteria and dust.

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Log-cabin strapping

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This drawing shows the arrangement of the adhesive strips. Note the oblique crossing or interweaving of the strips to form a firm dressing to the leg.

Log-cabin strapping is a type of bandaging used chiefly on the anterior of the leg, between the ankle and the knee, where the muscle and tissue over the bone are particularly thin. It is most useful in industry in treatment of employees with contusions and the ensuing hematoma.

Any wound involved is treated by the physician in the usual manner; then the log-cabin strapping is started well below the point of injury and carried well above it. Quarter-inch strips of adhesive are best suited to this purpose and strips from four to eight inches long, depending on the size of the area of the contusion, may be used. The strips are crossed one over the other. The first cross is started one inch from the end of the adhesive, applied well below the contused area, and continued in criss-cross strips to form an interwoven adhesive dressing.

This dressing should be applied as soon as possible after the injury. The quick application of the log-cabin strapping prevents swelling and edema of the periosteum, thereby equalizing the outer pressure against the inside edema and avoiding the dull ache which is experienced on this kind of injury, particularly if the patient is doing a standing job.

This dressing should be left in place for about three days unless there are indications to the contrary, which might be evidenced by pain or throbbing.